```
=> s atherosclerosis or arteriosclerosis
L2      226621 ATHEROSCLEROSIS OR ARTERIOSCLEROSIS

=> s tibolone or 5630-53-5/rn
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
'RN' IS NOT A VALID FIELD CODE
L3        1428 TIBOLONE OR 5630-53-5/RN

=> s l2 and l3
L4          49 L2 AND L3

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5          35 DUP REM L4 (14 DUPLICATES REMOVED)

=> s l5 and py<1997
   2 FILES SEARCHED...
L6          6 L5 AND PY<1997

=> d l6 1-6 ab bib kwic

L6   ANSWER 1 OF 6  EMBASE  COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB   This review summarizes recent data on the effects of endogenous and
     exogenous androgens, estrogens and progesterone on serum lipoproteins
     levels and composition in humans. Sex steroid hormones modulate serum
     lipoprotein metabolic mechanisms and influence atherosclerosis
     and coronary heart disease. In general, androgens lower HDL and raise LDL
     levels and Lp(a) thus promoting the atherogenic process. As it is true
     with estrogens, the lipoprotein effects of androgens are more pronounced
     with oral than with parenteral administration. Millions of women use oral
     contraception and postmenopausal women use more and more some form of
     hormone replacement therapy. The HDL-raising effect of estrogen
     replacement seems to be mediated by an increase in apoprotein AI
     production and not by a decrease in the clearance rate. Estrogens lower
     LDL levels by accelerating the rate of LDL catabolism which is due to an
     increase in the number of hepatic LDL receptors. They also improve
     endothelium-dependent vasodilatation which might be mediated by an
     antioxidant action of estrogens. These facts could explain well known
     cardioprotective effects of estrogens. Androgen progestins, especially
     older such as norgestrel, lower HDL and raise LDL thus diminishing or
     eliminating the benefits of estrogens on cardiovascular system while
newer
     progestins have a lesser effect on circulating lipoproteins.
AN   96168299  EMBASE
DN   1996168299
TI   [The effects of androgens and other sex steroid hormones on serum
     lipoproteins].
     UCINCI ANDROGENA I DRUGIH SPOLNIH HORMONA NA SERUMSKE LIPOPROTEINE.
AU   Reiner Z.
CS   Klinika za Unutarnje Bolesti, Klinicki Bolnicki Centar 'Rebro',
     Kispaticeva 12,Zagreb, Croatia
SO   Lijecnicki Vjesnik, (1996) 118/SUPPL. 1 (33-37).
     ISSN: 0024-3477  CODEN: LIVJA5
CY   Croatia
DT   Journal; Conference Article
FS   003     Endocrinology
     010     Obstetrics and Gynecology
     029     Clinical Biochemistry
     030     Pharmacology
```

037 Drug Literature Index
LA Serbo-Croatian
SL English; Serbo-Croatian
SO Lijecnicki Vjesnik, (1996) 118/SUPPL. 1 (33-37).
ISSN: 0024-3477 CODEN: LIVJA5
AB . . . and progesterone on serum lipoproteins levels and composition in humans. Sex steroid hormones modulate serum lipoprotein metabolic mechanisms and influence **atherosclerosis** and coronary heart disease. In general, androgens lower HDL and raise LDL levels and Lp(a) thus promoting the atherogenic process.. . .
CT Medical Descriptors:
*lipoprotein blood level
atherosclerosis
conference paper
female
hormone substitution
human
ischemic heart disease
lipoprotein metabolism
oral drug administration
*androgen: PD, pharmacology
*androgen: EC, endogenous compound
*estrogen: EC, endogenous compound
*estrogen: PD, pharmacology
*lipoprotein: EC, endogenous. . . compound
*sex hormone: PD, pharmacology
*sex hormone: EC, endogenous compound
danazol: PD, pharmacology
medroxyprogesterone acetate: PD, pharmacology
norethisterone: PD, pharmacology
norgestrel: PD, pharmacology
stanozolol: PD, pharmacology
testosterone: PD, pharmacology
tibolone: PD, pharmacology
RN (progesterone) 57-83-0; (danazol) 17230-88-5; (medroxyprogesterone acetate) 71-58-9; (norethisterone) 68-22-4; (norgestrel) 6533-00-2; (stanozolol) 10418-03-8, 302-96-5; (testosterone) 58-22-0; (**tibolone**) 5630-53-5

L6 ANSWER 2 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Menopause is the physiologic state that is a consequence of the cessation of ovarian function. A large number of vasomotor, psychological and gynecological symptons have been associated with menopause. Hormonal replacement therapy is effective in treating these conditions. The use of estrogens and progestins including dosages, routes of administration and their advantages and disadvantages are reviewed in this article. In addition, hormonal replacement therapy may reduce the risk of **atherosclerosis** and prevent the osteoporosis of climateric women. Hormonal therapy is associated with side effects but they do not contraindicate its use.
AN 95116094 EMBASE
DN 1995116094
TI [Hormonal replacement therapy in the climaterium].
TERAPIA DE SUSTITUCION HORMONAL EN EL CLIMATERIO.
AU Canto De Cetina T.E.
CS Depto. de Biol. de la Reproduccion, Ctro. de Invest. Reg. Dr. H. Noguchi, Universidad Autonoma, Calle 59 No. 490,Merida, Yucatan, Mexico
SO Revista de Investigacion Clinica, (1995) 47/1 (49-61).
ISSN: 0034-8376 CODEN: RICLAG
CY Mexico

DT Journal; General Review
FS 003 Endocrinology
010 Obstetrics and Gynecology
020 Gerontology and Geriatrics
030 Pharmacology
037 Drug Literature Index
LA Spanish
SL Spanish; English
SO Revista de Investigacion Clinica, (1995) 47/1 (49-61).
ISSN: 0034-8376 CODEN: RICLAG
AB . . . and their advantages and disadvantages are reviewed in this article. In addition, hormonal replacement therapy may reduce the risk of **atherosclerosis** and prevent the osteoporosis of climateric women. Hormonal therapy is associated with side effects but they do not contraindicate its. . .
CT Medical Descriptors:
*climacterium
*menopause
drug . . .
acetate: AE, adverse drug reaction
mestranol: AE, adverse drug reaction
norethisterone: AE, adverse drug reaction
norgestrel: AE, adverse drug reaction
progesterone: AE, adverse drug reaction
tibolone: AE, adverse drug reaction
RN (chlormadinone) 1961-77-9; (estradiol) 50-28-2; (estrone) 53-16-7; (ethinylestradiol) 57-63-6; (medroxyprogesterone acetate) 71-58-9; (mestranol) 72-33-3; (norethisterone) 68-22-4; (norgestrel) 6533-00-2; (progesterone) 57-83-0; (**tibolone**) 5630-53-5

L6 ANSWER 3 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Lipoprotein (a) has been implicated with an increased risk of **atherosclerosis** and cardiovascular disease. Recently, considerable progress has been made toward understanding the importance of genetics in the regulation of plasma levels of lipoprotein (a). However, the issue as to whether lipoprotein (a) levels should be treated is still debated and furthermore the possibilities to influence lipoprotein (a) levels remain limited. The potential clinical importance of Lipoprotein (a) has stimulated interest in the dietary and pharmacologic agents that affect this lipoprotein. At present, only a few of the existing therapeutic tools, such as nicotinic acid and estrogens, have been found to consistently affect lipoprotein (a).
AN 95071854 EMBASE
DN 1995071854
TI Diet and drug therapy for lipoprotein (a).
AU Berglund L.
CS Div of Preventive Medicine Nutrition, Dept Medicine, College of Physicians, Surgeons of Columbia University, 630 West 108th Street,New York, NY 10032, United States
SO Current Opinion in Lipidology, (1995) 6/1 (48-56).
ISSN: 0957-9672 CODEN: COPLEU
CY United Kingdom
DT Journal; General Review
FS 037 Drug Literature Index
038 Adverse Reactions Titles
LA English
SL English
SO Current Opinion in Lipidology, (1995) 6/1 (48-56).
ISSN: 0957-9672 CODEN: COPLEU
AB Lipoprotein (a) has been implicated with an increased risk of

atherosclerosis and cardiovascular disease. Recently, considerable progress has been made toward understanding the importance of genetics in the regulation of plasma. . .

CT Medical Descriptors:
*cardiovascular disease: DT, drug therapy
*cardiovascular disease: PC, prevention
*cardiovascular disease: TH, therapy
*diet therapy
apheresis
atherosclerosis: DT, drug therapy
atherosclerosis: PC, prevention
atherosclerosis: TH, therapy
cholesterol diet
clinical trial
double blind procedure
eskimo
fat intake
female
human
hypercholesterolemia
hyperlipoproteinemia: CO, complication
kidney disease
lipoprotein blood level
lipoprotein metabolism
low calory diet
male
medical genetics
mouse
non insulin dependent diabetes mellitus
nonhuman
postmenopause
primate
priority. . .
pharmacology
nicotinic acid: AE, adverse drug reaction
octreotide
pravastatin: DT, drug therapy
pravastatin: CB, drug combination
probucol: DT, drug therapy
stanozolol: DT, drug therapy
tamoxifen: PD, pharmacology
thyroid hormone
tibolone: DT, drug therapy
very low density lipoprotein: EC, endogenous compound

RN. . . 1404-04-2, 1405-10-3, 8026-22-0; (niceritrol) 5868-05-3; (nicotinic acid) 54-86-4, 59-67-6; (octreotide) 83150-76-9; (pravastatin) 81131-74-0;
(probucol) 23288-49-5; (stanozolol) 10418-03-8, 302-96-5; (tamoxifen) 10540-29-1; (**tibolone**) 5630-53-5

L6 ANSWER 4 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 92315534 EMBASE
DN 1992315534
TI The role of ovarian and testicular steroids in metabolism of lipids and in
atherogenesis.
AU Marek J.
CS III Interni Klinika, I Lekarska Fakulta, Univerzita Karlova, U nemocnice 1,128 21 Praha, Czechoslovakia
SO Vnitrni Lekarstvi, (1992) 38/9 (913-920).

ISSN: 0042-773X CODEN: VNLEAH
CY Czechoslovakia
DT Journal; Article
FS 003 Endocrinology
005 General Pathology and Pathological Anatomy
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
018 Cardiovascular Diseases and Cardiovascular Surgery
LA Czech
SL English; Czech
SO Vnitrni Lekarstvi, (1992) 38/9 (913-920).
ISSN: 0042-773X CODEN: VNLEAH
CT Medical Descriptors:
***atherosclerosis: SI, side effect**
***atherosclerosis: PC, prevention**
***atherosclerosis: DT, drug therapy**
*hypercholesterolemia: SI, side effect
*hypercholesterolemia: DT, drug therapy
*hypercholesterolemia: PC, prevention
*lipid metabolism
*lipoprotein metabolism
article
cardiovascular disease
drug effect
female
heart protection
hormone substitution
human
male
menopause
*estrogen: AE, adverse drug reaction
*estrogen: PD, pharmacology
*estrogen: DT, drug therapy
*gestagen: AE, adverse drug reaction
*tamoxifen: DT, drug therapy
*testosterone derivative: DT, drug therapy
***tibolone: DT, drug therapy**
allylestrenol
conjugated estrogen
estradiol
estriol
estrofem
levonorgestrel
lynestrenol
medroxyprogesterone acetate
mestranol
methyltestosterone: DT, drug therapy
norethisterone
norgestrel
tamoxifen citrate
RN (tamoxifen) 10540-29-1; (**tibolone**) 5630-53-5; (allylestrenol) 432-60-0; (estradiol) 50-28-2; (estriol) 50-27-1; (estrofem) 65296-29-9; (levonorgestrel) 797-63-7; (lynestrenol) 52-76-6; (medroxyprogesterone acetate) 71-58-9; (mestranol) 72-33-3; (methyltestosterone) 58-18-4;. .
.

L6 ANSWER 5 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AB Org OD 14 is a synthetic steroid which in animal bioassays displays oestrogenic as well as very weak androgenic-anabolic properties. Earlier

studies have shown that it alleviates oestrogen-deficiency symptoms and retards osteoporosis. OD 14 can be administered continuously with little effect on the endometrium. The aim of this study was to evaluate the effect of OD 14 on apolipoprotein A1 (Apo-A1), the major protein constituent of the high-density lipoprotein (HDL) fraction, as compared with that of oestradiol valerate (E2V) and a placebo. Twenty-two women, who had been oophorectomized when undergoing surgical treatment for stage IB or IIA cervical carcinoma, were given OD 14 2.5 mg/day, a placebo, and E2V 2 mg/day for a period of 6 wk in each case using a double-blind, cross-over method. Serum Apo-A1 was determined by electro-immunoassay after each treatment period. There was a marked decrease in Apo-A1 after OD 14 as compared with the levels seen after the placebo and E2V. This decrease is interpreted as evidence of a strong androgenic influence by OD

14. In epidemiological studies low levels of Apo-A1 have been associated with a higher incidence of **atherosclerosis** and cardiovascular disease. Long-term treatment with OD 14 might therefore be hazardous in this respect.

AN 85088669 EMBASE
DN 1985088669
TI Apolipoprotein A1 levels in oophorectomized women treated with Org OD 14, oestradiol valerate and a placebo.
AU Crona N.; Silfverstolpe G.; Enk L.; Samsioe G.
CS Department of Obstetrics and Gynecology, Sahlgrenska Hospital, Goteborg, Sweden
SO Maturitas, (1984) 6/4 (335-339).
CODEN: MATUDK
CY Netherlands
DT Journal
FS 037 Drug Literature Index
010 Obstetrics and Gynecology
030 Pharmacology
003 Endocrinology
LA English
SO Maturitas, (1984) 6/4 (335-339).
CODEN: MATUDK
AB . . . androgenic influence by OD 14. In epidemiological studies low levels of Apo-A1 have been associated with a higher incidence of **atherosclerosis** and cardiovascular disease. Long-term treatment with OD 14 might therefore be hazardous in this respect.
CT Medical Descriptors:
***atherosclerosis**
*cardiovascular disease
*lipid blood level
*drug therapy
*uterine cervix carcinoma
ovariectomy
priority journal
therapy
human
blood and hemopoietic system
female genital system
clinical article
endocrine system
cardiovascular system
*apolipoprotein a1
*estradiol valerate
*placebo
***tibolone**
RN (estradiol valerate) 979-32-8; (**tibolone**) 5630-53-5

L6 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2002 ACS

AB The effects of daily 2.5 mg/day doses of 7.alpha.,17.alpha.-hydroxy-7-methyl-19-norpregn-5(10)-en-20-yn-3-one (Org OD 14) on lipid metab., with particular ref. to high-d. lipoprotein (HDL)-related variables, were assessed in 14 healthy post-menopausal women after 12 and 36 mo of treatment. There were significant redns. in the following variables after both treatment periods: total phospholipids, total triglycerides, HDL-phospholipids and apolipoprotein A1. No changes were obsd. in total cholesterol or low-d. lipoprotein (LDL) cholesterol over the entire treatment period. A temporary decrease was obsd. in HDL-cholesterol after 12 mo, with a return to pretreatment values after 36 mo of treatment. The findings of this study clearly show that Org OD 14 has no adverse effects on the atherogenic variables, viz. LDL-cholesterol and triglycerides. Indeed , since the latter were lowered, its action is in fact beneficial. Moreover, its effect on HDL-cholesterol, the antiatherogenic variable, is only temporary. Although the compn. of HDL changes during Org OD 14 treatment (esp. as regards its cholesterol content), there is no evidence that reverse cholesterol transport is impaired.

AN 1990:509446 CAPLUS

DN 113:109446

TI Long-term effects of Org OD 14 on lipid metabolism in post-menopausal women

AU Kloosterboer, H. J.; Benedek-Jaszmann, L. J.; Kicovic, P. M.

CS Organon Sci. Dev. Group, Oss, 5340 BH, Neth.

SO Maturitas (**1990**), 12(1), 37-42
CODEN: MATUDK; ISSN: 0378-5122

DT Journal

LA English

SO Maturitas (**1990**), 12(1), 37-42
CODEN: MATUDK; ISSN: 0378-5122

IT **Atherosclerosis**
(Org OD 14 effect on lipid metab. in postmenopausal women in relation to)

IT **5630-53-5**, Org OD 14
RL: BIOL (Biological study)
(lipid metab. response to long-term administration of, in postmenopausal women)

=>